TWO CASES OF SYMMETRICAL NECROSIS OF THE CORTEX OF THE KIDNEYS ASSOCIATED WITH PUERPERAL ECLAMPSIA AND SUPPRESSION OF URINE.

By R. JARDINE and J. H. TEACHER.

Reprinted from The Journal of Pathology and Bacteriology.
Vol. XV. (1911).

Journal of Pathology and Bacteriology, Vol. XV.

TWO CASES OF SYMMETRICAL NECROSIS OF THE CORTEX OF THE KIDNEYS ASSOCIATED WITH PUERPERAL ECLAMPSIA AND SUPPRESSION OF URINE.

By R. Jardine and J. H. Teacher.

From the Pathological Laboratory, University of Glasgow.

(Plates XX.–XXII.)

Suppression of urine appears to be one of the rarer complications of the puerperium, and to depend upon a variety of lesions. Widespread necrosis of the cortex of the kidneys has been found in association with it in only about half a dozen cases, so that it must be an extremely rare lesion. The first case of which we are aware was that published by Rose Bradford and Lawrence in 1898, and there is little to be added to the account which they have given of the histology of the condition. Since then cases have been recorded by Griffith and Herringham, H. C. Lloyd, Klotz, and the first of the present cases was reported in another place by Jardine.

Clinically the cases present considerable variation in details, but all agree in the association of the puerperium, the presence of fits, and the absence of the usual uræmic phenomena. In most of the cases suppression of urine was not complete. Pathologically, the cases are practically identical. The leading features are symmetrical necrosis of the greater part of the cortex of the kidneys, which appears to be due to the presence of thrombi in the small interlobular and straight arteries of the cortex. In Lloyd's case the thrombi are described as agglutinations of red blood corpuscles; in the rest they were of the amorphous variety. The condition was clearly one of thrombosis—not of embolism. The cause of the thrombosis remains unknown.

The present cases are therefore worth recording on account of the rarity of the condition; but in addition our second case presents a feature which is very suggestive and seems to indicate an explanation of the peculiar localisation of the thrombosis. In the earlier part of the illness lividity resembling the local asphyxia of Raynaud's disease was well marked in the arms and face. The pulse was regular,

sometimes over 100, but at other times slow and very feeble,—barely perceptible at the wrist. It seems not improbable that the condition of spasm of the vessels existed in the internal organs, and particularly in the kidneys, which are usually regarded as apt to be affected by conditions affecting the skin. Spasm of the smaller branches of the renal arteries is therefore suggested as the determining cause of the thrombosis. The condition, if this explanation be accepted, would come into the category of symmetrical necroses similar to those which are seen at the extremities of the ears, nose, and limbs in certain cases of Raynaud's disease.

Case 1.

Clinical History.—Mrs. McC., 7-para, æt. 36, seven months pregnant, was admitted to the Glasgow Maternity Hospital on 11th March 1906, suffering from fits. These had commenced in the morning, and there had been seven or eight of them. A few days before, swelling of the legs and feet had been observed, and the day before admission the patient had complained of severe headache.

On admission the patient was unconscious, and very restless, with considerable œdema of the face, legs, and lumbar region. The uterus was soft and flaccid, and the os admitted a finger. The heart sounds were pure, and the pulse 110, of moderate tension. The urine became almost solid on being boiled.

Twelve fluid ounces of blood were drawn from the arm, and 2 pints of saline were transfused. The stomach was washed out, and a hot pack, a purgative and a dose of chloral and bromide were given. There were three more fits before midnight.

March 12.—The patient was semi-conscious, but did not reply to questions. It was afterwards discovered that she was stone-deaf. The urine contained 10 per 1000 albumin (Esbach) and over $1\frac{1}{2}$ per cent. of urea. There were granular tube casts but no blood in it. She passed 35 fluid oz. in twelve hours, *i.e.* from 9 p.m. on the 12th to 9 a.m. on the 13th.

March 13.—About 3.30 p.m. she delivered herself of a seven months' dead fœtus. A little urine was passed. There had been almost complete anuria from midday of the 13th. The urine obtained in the early morning showed only 2 per 1000 albumin (Esbach) and $1\frac{3}{4}$ per cent. of urea.

March 14.—At 3 p.m. the patient had a fit. The temperature was 98° F., and the pulse from 100 to 120. No urine was excreted. Saline infusion of 2 pints given.

March 15.—The patient was sick and vomited dark material. The mental condition was brighter, and she did not seem to suffer any inconvenience from the anuria. The temperature was 99° and the pulse 90. Patient took a good deal of fluid throughout.

March 16.—The skin showed a tinge of jaundice. The patient was quite bright mentally, and complained of hunger. At noon $\frac{1}{6}$ gr. of pilocarpine was given hypodermically, followed by a hot pack. This treatment, along with cupping or poulticing over the kidney, was repeated on subsequent days. About half a fluid ounce of urine was obtained by catheter. There was only a trace of albumin in it.

March 17.—The jaundice was a little deeper. One fluid drachm of urine was obtained by catheter. The pulse was still good, 80 to 90.

March 18.—Dr. Newman cut down on to the right kidney. The capsule was incised and stripped off one side of the kidney nearly to the hilum, and off

the other side slightly. An incision of about half an inch in length and one-third of an inch in depth was made into the kidney substance. There was little bleeding, and the appearance of the kidney was so strange that nothing more was done.

After the operation, patient became restless and complained of abdominal pain, but there were no convulsions. She died ten hours after the operation and about five and a half days from the commencement of the suppression of urine.

The post-mortem examination was made by Dr. Carstairs Douglas. There was some general œdema, and also slight ascites. The *liver* was of moderate size, pale and fatty. The *intestine* was normal. The *heart* was of moderate size, and fatty. The valves were healthy, and there were some ante-mortem clots in the right side. The *lungs* were congested and œdematous, and showed emphysema of the anterior borders. The *kidneys* and a portion of the liver were placed in formalin and sent to Dr. Teacher for examination. The kidneys were somewhat larger than normal. The capsule of the right kidney had been stripped off at the operation but replaced, and lay separated from the cortex by a layer of blood clot. The capsule of the other kidney was slightly thickened, but stripped readily. It was congested, and there were numerous small hæmorrhages between it and the surface of the kidney.

On section the kidneys (Plate XX. Fig. 1) presented an extremely striking appearance. They were congested, but the degree of contrast between the inner zone of the cortex and the medulla and their appearance were not far removed from normal. The remainder of the cortex, however, over practically the whole surface of the organ and in between the pyramids, was the seat of very profound changes, apparently of the nature of infarction. At all points there appeared to be a layer of normal cortex next the medulla, and in places there was some normal-looking tissue on the surface.

The infarcted tissue presented the usual dull, opaque, yellow appearance with congested and hæmorrhagic margins.

The pelves and ureters were normal. The walls of the larger blood vessels appeared to be slightly thickened, but there was no thrombosis or endarteritis visible to the naked eye. The blood vessels lying between the cortex and medulla were fairly prominent, and they also could be seen to be free from thrombosis.

Microscopic Examination.—Sections were prepared from portions of both kidneys, and mounted in series of some length with a view to following the small blood vessels which were seen to be occupied by thrombi. In the infarcted area all the structures were in a state of necrosis which was almost complete, nuclear staining as a rule having disappeared, although the outlines of the tubules and glomeruli were still quite distinct. The extent of the necrosis was even greater than seemed to be the case from the gross appearance of the organ, and it involved fully two-thirds of the entire cortex; but showed considerable irregularity. At most points there was a narrow zone close to the medulla which was not necrotic, and in places the living tissue extended almost to the surface. A very small amount of living cortex was also found scattered along the surface of the kidney. The capsule was slightly thickened but fibrous in character, and showed no sign of active inflammatory change. There was no difference between the two kidneys, the decapsulation having apparently produced no changes.

The kidney had not been perfectly normal, as there was a slight excess of interstitial connective tissue generally, and in certain areas there was a considerable amount of rather cellular tissue and swelling and catarrh and irregular dilatation of the tubules. Apparently there had been a slight subacute interstitial nephritis. In places there was considerable catarrh of the tubules with necrosis and shedding of the epithelium in the surviving

renal tissue, and this appeared to be a part of the necrotic process, because when there was sufficient cortex to allow of the tubules being quite clear of the infarction, the epithelium showed only slight cloudy swelling, or quite normal characters. There was no sclerosis of glomeruli.

The greater part of the infarcted area showed coagulation necrosis without any sign of reaction, but along the margins there was very pronounced infiltration by polymorpho-nuclear leucocytes. In parts these formed considerable collections round about the vessels and extending even into the medulla. Great numbers of them were also found in certain of the renal tubules. In the marginal zone there were also much congestion and hæmorrhage. Many of the glomeruli were gorged with blood; there was also some blood in the capsules and in a considerable number of tubules. In the border zone a number of tubules showed a peculiar staining reaction, the epithelium, the cells of which seemed to be loosened from one another, being stained of an intense blue colour with hæmatoxylin. The deep blue material was granular, but it was not calcareous. It did not retain Gram's stain. The condition appeared to be an advanced stage of degeneration, but not complete necrosis. Micro-organisms could not be found in sections stained by Gram's method or by thionin blue.

The cause of the condition appeared to be widespread thrombosis which had obstructed the smaller arteries of the cortex. The thrombi commenced a short distance on the medullary side of the infarction.

The larger vessels between the cortex and medulla and the greater part of the arterial arches and their accompanying veins were entirely free from thrombosis. For the most part they had been completely drained of blood. The thrombi in the arterial arches not infrequently occurred near the termination of these arteries where the vessel had become quite small, and the thrombus occupied the bend of the vessel and the commencement of the straight artery for a very short distance. Other thrombi, indeed the greater number, were situated in the arteriæ rectæ, usually near their origin from the arches.

The affected vessels varied in size from about 100 μ to rather over 200 μ in diameter. The thrombus was of the amorphous type, extremely dense and firm, and filled the vessel for a distance not much greater than two to four times its diameter. It tapered off into a thin layer close to the vessel wall extending some little distance in either direction. Distally the vessel, as a rule, was wider than at the point of dense thrombosis, and its cells and contents so completely necrotic that the nature of the contents could scarcely be made out. In a few cases it appeared to be closely packed with red blood corpuscles and a varying amount of irregular fibrinous material; in others it contained a mixed thrombus. There was a fairly well-marked arterio-sclerosis varying greatly in different vessels and affecting only the inner coat. The thickened intima consisted of firm fibrous tissue, and nothing was seen which in the least degree resembled acute or even subacute endarteritis. It was quite clear that the endarteritis could not be held to explain the occurrence of the thrombosis.

The veins in the neighbourhood of the dense thrombi, in many instances, were blocked by fairly firm thrombi of mixed character, which extended for a rather greater distance both towards the cortex and towards the hilum than those in the arteries. Farther out the contents were of loose structureless necrotic material. The type of thrombus suggested secondary formation, being much more cellular than that in the arteries.

Case 2.

Clinical History.—Mrs. R., æt. 22, eight months pregnant, was admitted to the Glasgow Maternity Hospital on 22nd January 1907, having had eight convulsive seizures during the preceding four hours.

Throughout pregnancy she had suffered from severe headaches, and during the last three months had complained of pain at the pit of the stomach.

Since the middle of December 1906, her feet had been swollen. During the last fourteen days, she frequently complained of specks before her eyes, and on the evening prior to admission became totally blind. On the evening of 21st January, she had a severe attack of vomiting, which was followed half an hour later by the first convulsive seizure. The latter lasted two or three minutes, and preceded a series of seven fits occurring at short intervals.

On admission her condition seemed to be critical. Her face presented a bloated appearance—the venules on the cheeks being injected and swelling of the eyelids and lips being well marked. The lower extremities were notably œdematous.

The pupils were moderately dilated and equal but reacted sluggishly to light. Temperature was 99° F.; pulse, 140, and very feeble and intermittent. She was unconscious, but was very restless and constantly ground her teeth. Urine and fæces were passed involuntarily. The fœtal heart sounds were not audible. She had four more fits in rapid succession. Chloroform was administered, bleeding and saline infusion performed, and a hot wet pack and a rectal injection of chloral and bromide were given. Urine drawn off by catheter showed a trace of blood, albumin in abundance, and granular tube casts.

January 23.—Patient had improved somewhat; she was semi-comatose but could be roused, and there had been no recurrence of fits. She delivered herself of a still-born child weighing $4\frac{3}{4}$ lb. Temperature ranged from 97° to 99°·6 F.; pulse from 88 to 120, and the respirations from 16 to 28 per minute. Twelve oz. of urine were passed during the last twenty-four hours, and a little was lost. The albuminuria registered a quarter above U mark, Esbach.

January 24.—Condition worse. Patient was sick and vomited some bilious material. Her face and extremities were cold and livid, not unlike a degree of Raynaud's disease. Temperature, 100°·4 to 96° F.; pulse, 100 to 74, very feeble (barely perceptible at the wrist), but regular and slow. Heart sounds somewhat distant but quite audible. The amount of urine passed was 8 oz., and a little was lost in two loose motions. Albuminuria, half above U mark, Esbach. Patient is less inclined to take fluids by the mouth.

January 25.—No improvement. Lividity and coldness of extremities still present. Vomiting continued. Temperature subnormal, 95° to 97° F.; respirations, 14 to 16. Degree of anuria more marked; only 1 oz. passed and very little lost. Albuminuria, half above U mark, Esbach. Motions loose and bloody.

January 26.—Circulation has improved a little, the lividity being less marked. Temperature, 96° to 98° F.; pulse, 70 to 92, of fuller volume; respirations, 18 to 20. Anuria still marked. Urine passed 3 oz., and a little lost. Albuminuria, three quarters above U mark, Esbach. Motions still loose and bloody.

January 27.—If anything the patient was a little more conscious and answered slowly when roused. The circulation had improved. Temperature, 95°·2 to 97° F.; pulse, 80 to 88; respirations, 17 to 28. Urine, very little passed and that lost. Motions loose and bloody.

January 28.—Patient drank more fluid, but otherwise her condition remained much the same. Temperature, 95°·8 to 97° F.; respirations, 15 to 26. Urine, half an ounce by catheter. Albuminuria, quarter above U mark, Esbach.

January 29.—No improvement. Temperature, 96° to 96°·4 F.; pulse, 92 to 108; respirations, 18 to 24. Urine, $1\frac{1}{2}$ oz. by catheter. Motions frequent, loose, but less tinged with blood. Drinking very little.

January 30.—No change; still semi-conscious. Temperature, 96°·4 to 98°·6 F.; pulse, 78 to 100; respirations, 18 to 24. Urine all lost. Motions frequent, loose, but blood practically absent.

January 31.—Temperature, 96° to 97° F.; pulse, 84 to 94; respirations, 18 to 22. Urine, 3 oz. Albuminuria, 8 per thousand, Esbach. Motions frequent, loose, dark green in colour, but not tinged with blood.

On 1st February, about 2.15 a.m., she suddenly became comatose—dying a quarter of an hour later at 2.30 a.m.

Post-Mortem Examination (*Abstract*).—The body was decidedly wasted; the mammæ were large and the nipples pigmented. No œdema of legs.

Thorax.—The mediastinal tissues were markedly œdematous. There were about 3 oz. of clear serous fluid in the pericardium, and a few ounces in each pleural sac. The heart was of normal size.

The cavities contained large agonal thrombi; and a large amount of fluid blood poured out when the large vessels were cut. In the apex of the left ventricle there was a thrombus about the size of a walnut firmly adherent to the wall. There was no infarction of the wall of the ventricle.

The lungs, apart from some œdema and hypostatic congestion, appeared to be normal.

Abdomen.—Contained about 20 oz. of clear serous fluid.

The liver and spleen presented normal healthy characters.

The stomach and small intestine presented nothing of note. The caput cæcum and ascending colon, and to a less degree the transverse colon, presented numerous hæmorrhagic and sloughing patches, corresponding to the points of exit of dilated veins. This appeared to be a dysenteric condition, which would account for the bleeding from the bowel noted during life.

Before proceeding to the removal of the kidneys all the large blood vessels in the abdomen were carefully examined, and not a trace of thrombosis was found except the normal thrombosis of the uterine sinuses.

The capsules of the kidneys were markedly injected; the kidneys showed a condition of infarction of almost the entire renal cortex. This was indicated by yellow colour, avascularity, and firm consistence. The necrosis appeared to extend right to the capsule; internally it stopped short of the medulla, leaving a thin layer of living cortex next the boundary zone. The necrosed tissue formed a remarkably even layer about 4 to 5 mm. thick, enclosing the whole organ and dipping down between the pyramids wherever there was cortex. It was bounded by a narrow line of intense congestion.

The pyramids appeared to be normal. The ureters were somewhat wider than normal, but there had been no obstruction. The bladder was normal and empty.

The suprarenal bodies appeared to be normal.

Examination of the head was not permitted.

Microscopic Examination.—Sections were prepared from the kidneys, the renal vessels in the hilum, the heart with the adherent thrombus, liver, spleen, great intestine, suprarenal and a lymphatic gland which lay near the left kidney.

The liver showed fairly pronounced fatty infiltration and some cloudy swelling, but no thrombosis or focal necrosis.

The spleen was congested; the Malpighian bodies appeared to be small and the pulp rather cellular.

In the lymphatic gland the lymphoid masses appeared small relatively to the lymph paths, which appeared very wide and full of oval cells. There was no marked congestion and no necrosis.

The suprarenal showed pronounced cloudy swelling affecting both cortex and medulla. There were also patches in which the cells were separated from one another as if in an early stage of necrosis, and there were a few areas in which they were quite definitely necrotic. These were seen both in the cortex and in the medulla. They were very small. The blood vessels were intensely gorged with blood, but no definite thrombosis was found, and the little patches of necrosis did not suggest infarction.

The hæmorrhagic patches in the great intestine were areas of somewhat superficial necrosis of the mucous membrane on the crests of the folds. The submucous connective tissue was thickened and the whole patch was intensely congested, and in some instances there had been considerable extravasation of blood. Thrombosis of the vessels was seen only in the necrotic centres. The larger vessels of the intestinal wall and those of the mesentery were packed with red blood corpuscles.

Heart.—Sections were prepared at two levels transversely to the left ventricle, and a strip about two inches in length was taken longitudinally extending right to the apex. All three sections traversed the thrombus. One portion of the thrombus, situated about 3 cms. from the apex, had the appearance of a white globular thrombus such as is found in the auricular appendices in chronic cardiac disease. The rest, while of somewhat similar character, was much softer and more mixed with red blood. It was clearly, however, a slowly formed thrombus. To a considerable extent it lay in contact with the endocardium, but distinctly free from it. In other parts, however, the structures were completely incorporated, so that it was impossible to say where one ended and the other began. In many parts the edge of the thrombus showed a mixture of fibrin and connective tissue. The connective tissue was somewhat cellular and in places contained numerous wide capillaries distended with blood. Processes of the thrombus penetrated deeply into the muscle between the columnæ carneæ. In a few instances small thrombosed vessels were seen at some little depth in the heart wall, and throughout the fibrin-infiltrated tissue there were extravasations of blood. The larger branches of the coronary vessels showed no sign of disease. Some of the fibres of the myocardium next the thrombus showed degenerative changes, but there was nothing resembling infarction. It appears, therefore, that the thrombosis was not secondary to changes in the myocardium but was due, in all probability, to the stagnation of the circulation and other obscure conditions which lead to the formation of similar thrombi in various diseases.

Kidney.—The condition of the kidney (Plate XXI. Fig. 2) was very similar to that in the first case, but the infarction of the cortex was more uniform in extent and character. There was practically no living tissue under the capsule, and the layer of necrotic tissue was very regular and uniform in thickness. It involved at least two-thirds of the cortex. The comparative amount of necrosis in the two specimens cannot be accurately estimated by reference to the drawings. In the first specimen most of the zone which appears only congested is really necrotic, and the condition is more nearly coextensive with that in No. 2 than would be inferred from the gross character of the organ. The degree of completeness of the necrosis in the infarcted area is very similar in the two cases.

There is at most points a thin layer of living cortex between the necrotic tissue and the medulla. The margin of the necrosis is marked by a zone of intense congestion and the extravasation of blood.

The vessels at the hilus of the kidney appeared to be normal. Careful dissection was made of branches as far as they could be traced, and neither thrombus nor embolus could be found. Microscopic examination showed further that the vessels at the junction of the cortex and medulla and the straight vessels of the pyramids were also free from thrombosis.

The smaller arteries of the cortex presented a condition identical with that seen in the preceding specimen, being occupied by dense white amorphous thrombi. (Plate XXII. Figs. 3 and 4.) The extent of vessel occupied by this material varied somewhat. The thrombus commenced usually a little below the edge of the infarction. The size of the affected arteries varied from about 100 to 200 micromillimetres in diameter, most commonly nearer to the lower figure. Their walls were clearly muscular. There was very slight endarteritis.

Dense thrombi were found only in the arteries. A number of them were followed through serial sections, and it was found that the dense thrombi were very short, and beyond them the arteries were packed with red blood corpuscles and a varying amount of mixed thrombus. The tubules and glomeruli in the infarct were still recognisable by their characteristic outlines; but the nuclear staining was completely lost.

The condition of the accompanying veins differed from that in Case 1. It was frequently difficult to recognise the veins, as they were collapsed and empty. In other instances they contained abundance of red blood corpuscles or loose mixed thrombus. There was very little evidence of reaction at the margins of the infarct as a whole; but in a few places, especially near the larger vessels, there was considerable leucocytic infiltration.

The principal feature of this case is the condition of the kidney,—the lesions in the heart, intestines, and suprarenal being probably of secondary importance. It is a pity that permission to examine the head was refused, as this case differed from most of the others in presenting cerebral symptoms.

In attempting to explain the condition under discussion it is assumed that, as the fits preceded the anuria by a considerable interval of time, the cause or causes which gave rise to puerperal eclampsia were probably in operation prior to the occurrence of the necrosis of the kidney. At the same time it is questionable whether the present cases should be regarded as ordinary puerperal eclampsia. The ordinary lesions in the liver were absent.

The possibility that the symmetrical necrosis might be primary and the thrombosis secondary was considered and rejected for reasons which will be given below. This view has recently been put forward by Parkes Weber with reference to cases like those now under consideration.

In puerperal eclampsia the most constant lesions are focal necrosis, hæmorrhages and thrombosis of arteries and veins occurring in many parts of the body but most strikingly in the liver, lungs, and kidneys. In the last Schmorl frequently saw widespread necrosis of the epithelium of the tubules of the cortex, and in a few cases there were small infarctions. Schmorl regarded some of the infarctions as embolic in origin, but others as due to thrombosis of the smaller arteries. He considered and rejected the idea that the parenchymatous lesion might be primary and the thrombosis secondary. It must be noted that the lesions described by Schmorl and those in the present cases are very different. Whether they may or may not be related we are not at present prepared to say.

Against the idea that the symmetrical necrosis of the cortex is primary and the thrombosis secondary are the following facts:—

1. The necrosis is not complete in any of the cases. There is usually some surviving cortical tissue under the capsule, and a considerable amount between the infarct and the medulla. The necrosis is confined to the area of distribution of the thrombosed vessels.

2. The thrombi in the arteries and the veins are entirely different. The dense thrombi are confined to the arteries. The veins in one of our cases show the effects of intense congestion, in the other they are empty; in both there is secondary thrombosis, principally within the infarcted area, and probably related to necrosis of their walls. The *arterial* walls in the neighbourhood of the thrombi on the other hand are living and fairly healthy.

3. The appearance and structure of the affected renal tissue is exactly that of an anæmic infarct, such as is produced by an embolism, and we are of the opinion that the condition is of this nature although of different causation, namely, due to thrombosis.

The fact that the necrosis of the cortex is not total, but that it corresponds in extent to the distribution of the thrombosis, seems to indicate that this condition was the ultimate cause of the infarction. Spasm of the vessels was probably the determining cause of the thrombosis.

Spasm of the blood vessels has been held, probably with justice, to be a contributory cause of the necrosis of the renal epithelium in eclampsia and other toxic states on the analogy of what is known to result from experimental temporary obstruction of the renal artery, but, as already pointed out, that condition is not the same as the infarction under discussion.

The pathology of the gangrene of Raynaud's disease is obscure, if we except cases in which large portions of the limbs become gangrenous owing to endarteritis obliterans, embolism, or thrombosis of a large artery. The symmetrical necrosis of the renal cortex, however, should be compared with the gangrene affecting symmetrically only small portions of the extremities; but, according to Osler (1909), the causation of this is not always the same. Thrombosis and endarteritis have been found, but "it is well to insist upon the fact that the most advanced necrosis may occur as a consequence of spasm in vessels apparently healthy."

This statement seems to apply to cerebral softening due to spasm of the vessels as well as to Raynaud's disease. In some cases, however, thrombosis has been recorded. One of the most recent is that recorded by Allan and Wilson, in which gangrene of the fingers and nose was associated with thrombi in the digital arteries and widespread softening of the brain with thrombosis of minute arterioles. The writers concluded that arterial spasm was the determining cause of the thrombosis.

In conclusion, we are of opinion that there are several factors which must be considered in attempting to explain the lesion of the kidneys and its symmetrical character. Doubtless, the renal epithelium had suffered from the presence of toxic material and probably, having regard to the history of the second case, from deprivation of blood; but the condition of infarction involves not

only the structures which are known to be principally affected by these conditions, but also the glomeruli and interstitial tissues. To explain it some other factor must be brought in, and this was undoubtedly the thrombosis in the smaller renal arteries. Hæmorrhage, weakness of the circulation, and the puerperal state may all have helped to bring about the thrombosis, but we are of opinion that the ultimate cause of it was spasmodic contraction of the renal vessels. This gives the most satisfactory explanation of the symmetry of the lesion.

REFERENCES.

Allan, G. A., and G. Haswell Wilson	*Glasg. Med. Journ.*, 1910, vol. lxxiv. p. 25.
Bradford, Rose, and Lawrence	*Journ. Path. and Bacteriol.*, Cambridge, 1898, vol. v. p. 197.
Griffith and Herringham	*Ibid.*, 1906, vol. xii. p. 237.
Jardine, R.	*Journ. Obst. and Gynæc. of British Empire*, London, 1906, vol. x. p. 32.
Klotz, Oscar	*Am. Journ. Obst.*, N.Y., 1908, vol. lviii. p. 619.
Lloyd, H. C.	*Lancet*, London, 1906, vol. i. p. 146.
Osler	in Osler and McCrae's "System of Medicine," London, 1909, vol. vi. p. 625.
Schmorl	"Puerperal Eclampsia," Leipzig, 1893.
Weber, Parkes	*Lancet*, London, 1909, vol. i. p. 601.

DESCRIPTION OF PLATES.

Plate XX.

Fig. 1.—The kidney from Case 1, showing the irregular distribution of the necrosis (infaretion). Cf. text, p. 139.

Plate XXI.

Fig. 2.—The kidney from Case 2, showing the extremely complete and regular necrosis (infaretion) of the cortex.

Plate XXII.

Fig. 3.—Thrombosed artery and vein from the edge of the infaret in Case 1, showing the short, dense amorphous plug in the artery and thrombus of red or mixed character in the vein. There is a great deal of hæmorrhage between the tubules, which are nearly all necrotic. (× 80.)

Fig. 4.—Thrombosed artery from the edge of the infaret in Case 2. Note how sharply defined the infaretion is, but the position of its margin is similar to that in Case 1. The vein is not recognisable. Most of the red thrombus has fallen out of the distal portion of the artery. (× 80.)

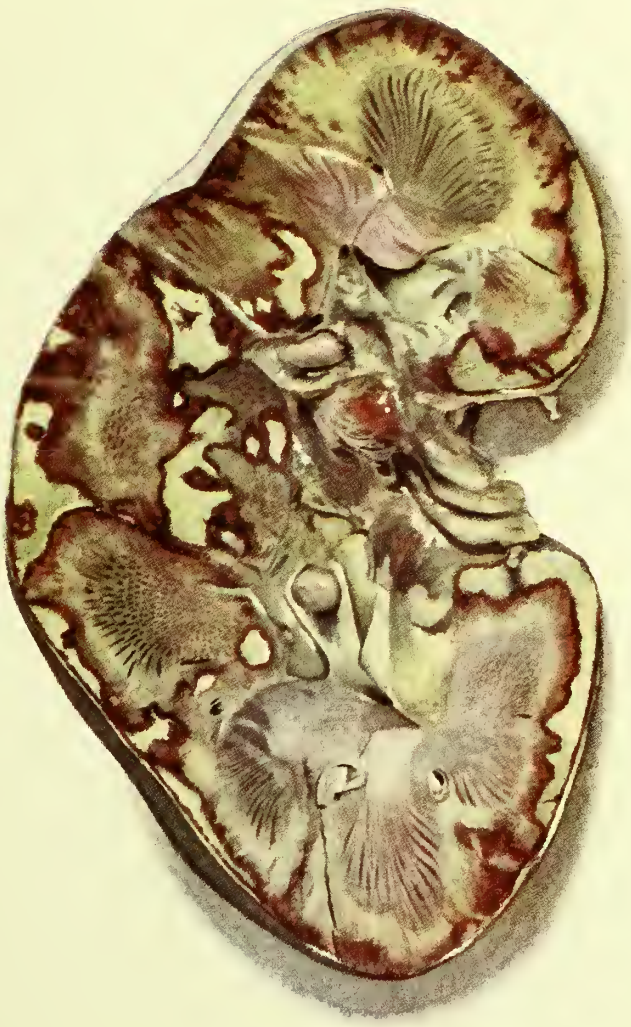

Fig. 1.

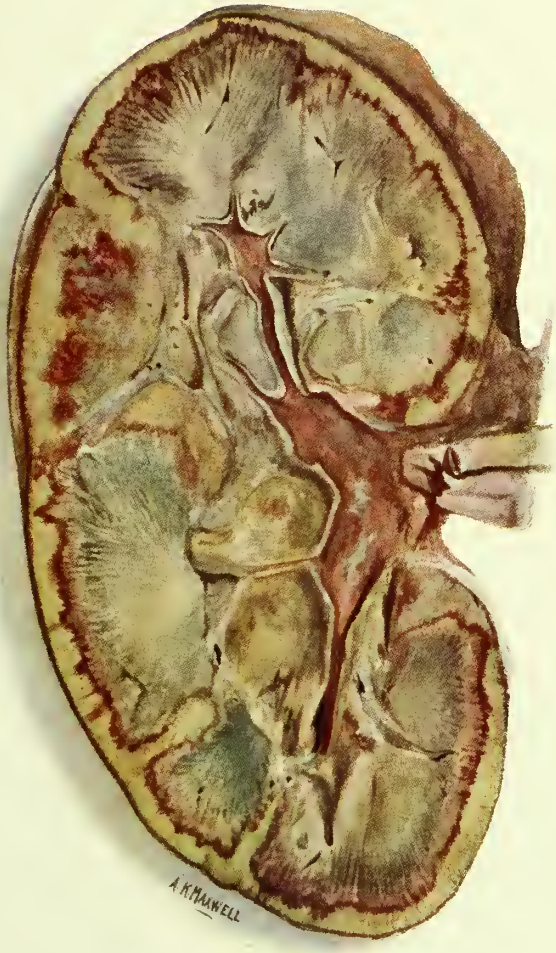

FIG. 2.

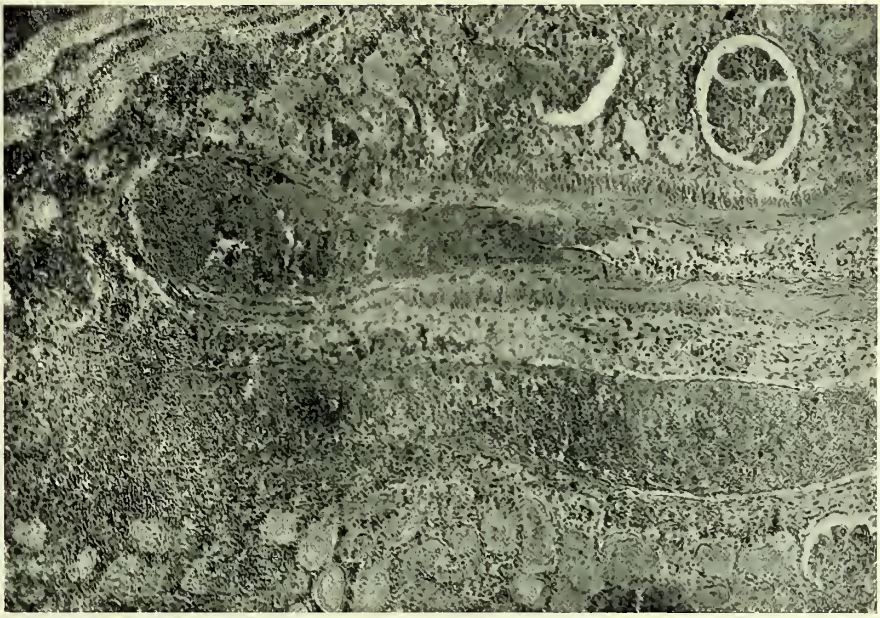

FIG. 3.

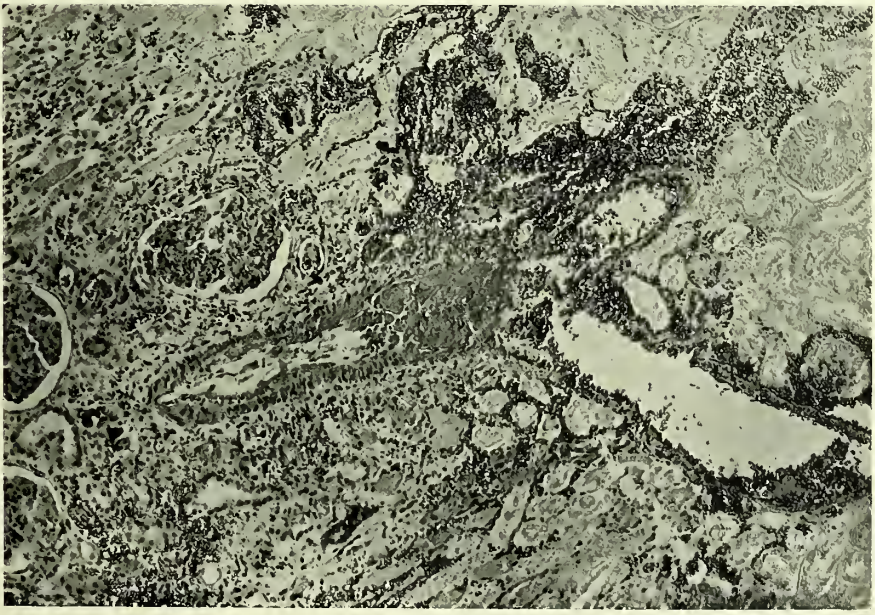

FIG. 4.